EDITORIALS

WJM Welcomes Nevada

WITH this issue THE WESTERN JOURNAL OF MEDICINE becomes the official journal of the Nevada State Medical Association and its members join the membership of the California and Idaho Medical Associations in receiving this journal as a benefit of membership in these associations. The Nevada and Idaho editions of WJM may from time to time contain additional material of particular interest to the members of those state associations but which may be of less interest to members in another state. Contributions from all three states are welcome and are given equal consideration by the Editorial Board—which is broadly representative of all the western states.

It is just three years since *California Medicine* entered its chrysalis and emerged as THE WESTERN JOURNAL OF MEDICINE. Long (since 1902) the voice of medicine in California, it aspired to reflect the vitality and achievements in science, education and practice which are the marks of medicine in the western United States; in a stronger presence in the medical literature which records and shares advances and progress with the medical community in the nation and indeed the world.

The WESTERN JOURNAL seeks to offer a variety of fare for its readers and to be truly a useful journal. Besides material useful to practitioners, it seeks also to have something of interest for students, academicians, researchers and even for the political leaders of our profession. The circulation is growing, with subscribers in every state of the Union and not a few from abroad. The editors, the Editorial Board and many others are dedicated to making this journal a regional instrument of physician education and continuing education in the West, and also to make it an increasingly distinguished journal on the national scene.

We welcome the Nevada State Medical Association to the WJM family of associations and its members to our readership. We look forward to a long and productive association which should be of great benefit to all concerned.

—MSMW

Aminoglycoside Nephrotoxicity

CONFIRMATION IN MAN of findings in animal models relevant to human disease or therapy is exemplified in the case report in this issue by Bennett and co-workers. They are almost the first to show the proximal renal tubular abnormalities due to aminoglycosides previously shown in rats and are the first to document the persistence of these changes as long as six weeks after stopping aminoglycoside therapy. These agents, notably gentamicin, are the most widely used antibiotics in hospitals except for the penicillins and cephalosporins and contribute very materially to the management of Gram-negative bacterial sepsis. All of them (streptomycin, kanamycin, gentamicin, tobramycin, amikacin, neomycin) have two major drawbacks—the development of high level bacterial resistance and a margin between safety and toxicity far less salubrious than in the case of the penicillins and cephalosporins. Fortunately, early gloomy predictions for a high prevalence of bacterial resistance have been confined largely to small population groups (such as burn units, newborn nurseries and urology wards) and early concern for toxicity has been modified as methods have been appreciated for calculating dosage, monitoring blood levels and detecting early evidence of impaired renal function.

As soon as the first two aminoglycosides, neomycin and streptomycin, were introduced to clinical usage in the mid-1940's, nephrotoxicity was evident—serious and frequent with neomycin, rare and almost confined to patients with preexisting renal impairment with streptomycin. Early pharmacologic studies established the linear relationship between creatinine or inulin clearance and aminoglycoside excretion and most physicians probably regard their elimination as a simple filtration process without participation of the renal tubule. Insufficient attention may have been paid to the pathology of aminoglycoside toxicity in animals which identified the proximal renal tubular cells as a primary target, and the ability of diuretic agents which act primarily on tubular cell function such as furosemide and ethacrynic acid to enhance aminoglycoside nephro-